

* * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 11:59:37 ON 15 JAN 2005

=> FIL MEDLINE SCISEARCH EMBASE BIOSIS

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|----------------------|------------------|---------------|
| FULL ESTIMATED COST | 0.21 | 0.21 |

FILE 'MEDLINE' ENTERED AT 11:59:56 ON 15 JAN 2005

FILE 'SCISEARCH' ENTERED AT 11:59:56 ON 15 JAN 2005
Copyright (c) 2005 The Thomson Corporation.

FILE 'EMBASE' ENTERED AT 11:59:56 ON 15 JAN 2005

COPYRIGHT (C) 2005 Elsevier Inc. All rights reserved.

FILE 'BIOSIS' ENTERED AT 11:59:56 ON 15 JAN 2005

Copyright (c) 2005 The Thomson Corporation.

=> s RECEPTOR (P) (LIGAND OR AGONIST OR ANTAGONIST OR COMPOUND) (P) (SCREEN? OR BIND? OR ASSAY) and orphan

3 FILES SEARCHED...

L1 2546 RECEPTOR (P) (LIGAND OR AGONIST OR ANTAGONIST OR COMPOUND) (P)
(SCREEN? OR BIND? OR ASSAY) AND ORPHAN

=> s l1 and py<=1998

2 FILES SEARCHED...

L2 912 L1 AND PY<=1998

=> dup rem l2

PROCESSING COMPLETED FOR L2

L3 284 DUP REM L2 (628 DUPLICATES REMOVED)

=> s l3 and (gpcr or g adj protein?coupled adj receptor)

'?' TRUNCATION SYMBOL NOT VALID WITHIN 'PROTEIN?COUPLED'

The truncation symbol ? may be used only at the end of a search term. To specify a variable character within a word use '!!', e.g., 'wom!n' to search for both 'woman' and 'women'. Enter "HELP TRUNCATION" at an arrow prompt (>) for more information.

=> s l3 and (gpcr or g (w) protein)

L4 33 L3 AND (GPCR OR G (W) PROTEIN)

=> d 1-33 bib ab

L4 ANSWER 1 OF 33 MEDLINE on STN

AN 2004192058 IN-PROCESS Full-text

DN PubMed ID: 12671309

TI The Potential Role of the Transcription Factor RZR/ROR as a Mediator of Nuclear Melatonin Signaling.

AU Wiesenbergs I; Missbach M; Carlberg C

CS Pharma Research, NOVARTIS, Basel, Switzerland.

SO Restorative neurology and neuroscience, (1998) 12 (2-3) 143-50.
Journal code: 9005499. ISSN: 0922-6028.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS IN-DATA-REVIEW; IN-PROCESS; NONINDEXED

ED Entered STN: 20040417

Last Updated on STN: 20041219

AB The pineal gland hormone melatonin is well known as a regulator of circadian rhythmicity, but has also other functions in the central nervous system as well as in the periphery including the maturation of neurons and the regulation of cellular growth and differentiation. Three mechanisms of the hormone's action are currently discussed: a membrane signaling pathway, a nuclear signaling pathway and a receptor-independent radical scavenging function. Melatonin membrane receptors are seven transmembrane receptors and mediate their functions through a G-protein-coupled second messenger pathway. Nuclear melatonin signaling seems to be mediated via the transcription factor RZR/ROR, which is an orphan member of the nuclear receptor superfamily. The widespread distribution of the a-subtype of RZR/ROR suggests that this receptor may be an important mediator of those effects of melatonin that can not be explained by membrane receptors or radical scavenging. Interestingly, natural RZR/ROR^a "knock-out" mice (staggerer) show severe defects in the development of cerebellar Purkinje cells, a reduced body weight and immunological defects. RZR/ROR binds as a monomer to DNA, but also forms homodimers on appropriate binding sites. Natural RZR/ROR binding sites have been identified in the regulatory regions of many genes. 5-lipoxygenase, p21WAF1/CIP1, apolipoprotein A-1, N-myc and Purkinje cell protein 2 may be functionally important target genes. On some of these binding sites RZR/ROR competes with other members of the nuclear receptor superfamily (e.g., COUP-TF, RAR and Rev-ErbA) indicating a cross-talk between these transcription factors RZR/ROR often shows in transient transfection assays a high constitutive, i.e. ligand-independent activity. However, under conditions of low constitutive activity a significant and specific stimulation of RZR/ROR by low nanomolar concentrations of melatonin and structurally novel class of thiazolidinediones (lead structure: CGP52608) has been observed. Taken together, the effects of melatonin on transcriptional regulation clearly depend on the expression of RZR/ROR and support the concept that the receptor is a mediator of nuclear melatonin signaling.

L4 ANSWER 2 OF 33 MEDLINE on STN
AN 1999098533 MEDLINE Full-text
DN PubMed ID: 9884064
TI Orphan G-protein-coupled receptors: the next generation of drug targets?.
AU Wilson S; Bergsma D J; Chambers J K; Muir A I; Fantom K G; Ellis C; Murdock P R; Herrity N C; Stadel J M
CS SmithKline Beecham Pharmaceuticals, Harlow, Essex.
SO British journal of pharmacology, (1998 Dec) 125 (7) 1387-92.
Ref: 25
Journal code: 7502536. ISSN: 0007-1188.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199904
ED Entered STN: 19990413
Last Updated on STN: 20000303
Entered Medline: 19990401
AB The pharmaceutical industry has readily embraced genomics to provide it with new targets for drug discovery. Large scale DNA sequencing has allowed the identification of a plethora of DNA sequences distantly related to known G protein-coupled receptors (GPCRs), a superfamily of receptors that have a proven history of being excellent therapeutic targets. In most cases the extent of sequence homology is insufficient to assign these 'orphan' receptors

to a particular receptor subfamily. Consequently, reverse molecular pharmacological and functional genomic strategies are being employed to identify the activating ligands of the cloned receptors. Briefly, the reverse molecular pharmacological methodology includes cloning and expression of orphan GPCRs in mammalian cells and screening these cells for a functional response to cognate or surrogate agonists present in biological extract preparations, peptide libraries, and complex compound collections. The functional genomics approach involves the use of 'humanized yeast cells, where the yeast GPCR transduction system is engineered to permit functional expression and coupling of human GPCRs to the endogenous signalling machinery. Both systems provide an excellent platform for identifying novel receptor ligands. Once activating ligands are identified they can be used as pharmacological tools to explore receptor function and relationship to disease.

L4 ANSWER 3 OF 33 MEDLINE on STN
AN 1999084057 MEDLINE Full-text
DN PubMed ID: 9866825
TI Effects of newly isolated opioid peptides on G-protein activation: usefulness of [35S] GTP gamma S binding study and its practical application.
AU Narita M
CS Department of Anesthesiology, Medical College of Wisconsin, Milwaukee 53226, USA.
SO Nihon shinkei seishin yakurigaku zasshi = Japanese journal of psychopharmacology, (1998 Aug) 18 (4) 107-16. Ref: 58
Journal code: 9509023. ISSN: 1340-2544.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA Japanese
FS Priority Journals
EM 199902
ED Entered STN: 19990316
Last Updated on STN: 20000303
Entered Medline: 19990226
AB Recent cloning and expression studies have revealed that the opioid mu-, delta-, kappa- and orphan receptors are seven-transmembrane domain receptors whose actions are mediated through activation of guanine nucleotide binding protein (G-protein). The activation of G-proteins by the opioid receptor can be measured by assessing agonist stimulation of membrane binding of the non-hydrolyzable analog of guanosine triphosphate (GTP), guanosine-5'-O-(3-[35S] thio) triphosphate ([35S] GTP gamma S). Our recent data suggest that 1) the level of spinal mu-, delta-, kappa- and orphan-receptor agonist-stimulated [35S] GTP gamma S binding closely parallels that of receptor binding densities, 2) the neuroanatomical distribution of opioid agonist-stimulated [35S] GTP gamma S binding relates to receptor binding distribution, 3) newly isolated opioid peptides, endomorphin-1 and -2, can activate G-proteins by specific stimulation of mu-receptors and act as partial agonists with moderate catalytic efficacies, 4) mu-receptor densities could be rate-limiting steps in the G-protein activation by mu-agonists in the spinal cord region. In conclusion, opioid agonist-stimulated [35S] GTP gamma S binding can provide a functional method to localize receptors not only by their ability to bind ligands, but also according to their ability to activate an intracellular signal transducer.

AN 1998341734 MEDLINE Full-text
DN PubMed ID: 9678594
TI Functional genomics: the search for novel neurotransmitters and neuropeptides.
AU Civelli O
CS Department of Pharmacology, University of California, Irvine 92697-4625,
USA.. ocivelli@uci.edu
SO FEBS letters, (1998 Jun 23) 430 (1-2) 55-8. Ref: 40
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980903
Last Updated on STN: 19980903
Entered Medline: 19980825
AB Functional genomics can be defined as the search for the physiological role of a gene for which only its primary sequence is known. Most of the genes encoding proteins containing seven hydrophobic stretches code for G protein-coupled receptors (GPCRs). Although many of these have been shown to interact with known natural ligands, several bind ligands which have not been thus far isolated. These are the so-called orphan GPCRs. As an example of functional genomics, an 'orphan receptor strategy' has been developed to identify the natural ligands of orphan GPCRs. The application of this strategy is bound to revolutionize our understanding of the diversity of the primary messengers which modulate synaptic transmission. This review discusses the basic concepts and some of the particular problems associated with the orphan receptor strategy. The strategy's potential is exemplified by its successes which culminated in the discovery of the neuropeptides 'orphanin FQ/nociceptin' and 'orexins/hypocretins'. The steps that led to the characterization of these neuropeptides are discussed as are some of the further studies that have addressed the roles of these neuropeptides. To conclude, some of the implications of the application of the orphan receptor strategy are discussed.

L4 ANSWER 5 OF 33 MEDLINE on STN
AN 1998264639 MEDLINE Full-text
DN PubMed ID: 9603476
TI ChemR23, a putative chemoattractant receptor, is expressed in monocyte-derived dendritic cells and macrophages and is a coreceptor for SIV and some primary HIV-1 strains.
AU Samson M; Edinger A L; Stordeur P; Rucker J; Verhasselt V; Sharron M;
Govaerts C; Mollereau C; Vassart G; Doms R W; Parmentier M
CS IRIBHN, Universite libre de Bruxelles, Belgium.
NC AI-35383 (NIAID)
AI-38225 (NIAID)
AI-40880 (NIAID)
SO European journal of immunology, (1998 May) 28 (5) 1689-700.
Journal code: 1273201. ISSN: 0014-2980.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
OS GENBANK-Y14838
EM 199806
ED Entered STN: 19980611

Last Updated on STN: 19980611
Entered Medline: 19980603

AB Leukocyte chemoattractants act through a rapidly growing subfamily of G protein-coupled receptors. We report the cloning of a novel human gene encoding an orphan receptor (ChemR23) related to the C3a, C5a and formyl Met-Leu-Phe receptors, and more distantly to the subfamilies of chemokine receptors. ChemR23 transcripts were found to be abundant in monocyte-derived dendritic cells and macrophages, treated or not with LPS. Low expression could also be detected by reverse transcription-PCR in CD4+ T lymphocytes. The gene encoding ChemR23 was assigned by radiation hybrid mapping to the q21.2-21.3 region of human chromosome 12, outside the gene clusters identified so far for chemoattractant receptors. Given the increasing number of chemoattractant receptors used by HIV-1, HIV-2 and SIV as coreceptors, ChemR23 was tested in fusion assays for potential coreceptor activity by a range of viral strains. None of the tested HIV-2 strains made use of ChemR23 as a coreceptor, but several SIV strains (SIVmac316, SIVmac239, SIVmac17E-Fr and SIVsm62A), as well as a primary HIV-1 strain (92UG024-2) used it efficiently. ChemR23 therefore appears as a coreceptor for immunodeficiency viruses that does not belong to the chemokine receptor family. It is also a putative chemoattractant receptor relatively specific for antigen-presenting cells, and it could play an important role in the recruitment or trafficking of these cell populations. Future work will be required to identify the ligand(s) of this new G protein-coupled receptor and to define its precise role in the physiology of dendritic cells and macrophages.

L4 ANSWER 6 OF 33 MEDLINE on STN
AN 1998244535 MEDLINE Full-text
DN PubMed ID: 9585136
TI Molecular mechanisms of G protein-coupled receptor desensitization and resensitization.
AU Ferguson S S; Zhang J; Barak L S; Caron M G
CS John P. Robarts Research Institute and Department of Physiology,
University of Western Ontario, London.
SO Life sciences, (1998) 62 (17-18) 1561-5. Ref: 22
Journal code: 0375521. ISSN: 0024-3205.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199805
ED Entered STN: 19980609
Last Updated on STN: 20000303
Entered Medline: 19980526
AB Beta-arrestin proteins play a dual role in regulating G protein-coupled receptor (GPCR) responsiveness by contributing to both receptor desensitization and internalization. Recently, beta-arrestins were also shown to be critical determinants for beta2-adrenergic receptor (beta2AR) resensitization. This was demonstrated by overexpressing wild-type beta-arrestins to rescue the resensitization-defect of a beta2AR (Y326A) mutant (gain of function) and overexpressing a dominant-negative beta-arrestin inhibitor of beta2AR sequestration to impair beta2AR dephosphorylation and resensitization (loss of function). Moreover, the ability of the beta2AR to resensitize in different cell types was shown to be dependent upon beta-arrestin expression levels. To further study the mechanisms underlying beta-arrestin function, green fluorescent protein was coupled to beta-arrestin2 (beta arr2GFP), thus allowing the real-time visualization of the agonist-dependent trafficking of beta-arrestin in living cells. Beta arr2GFP

translocation from the cytoplasm to the plasma membrane proceeded with a time course, sensitivity and specificity that was indistinguishable from the most sensitive second messenger readout systems. Beta arr2GFP translocation was GRK-dependent and was demonstrated for 16 different ligand-activated GPCRs. Because beta-arrestin binding is a common divergent step in GPCR signalling, this assay represents a universal methodology for screening orphan receptors, GRK inhibitors and novel GPCR ligands . Moreover, beta arr2GFP provides a valuable new tool to dissect the biological function and regulation of beta-arrestin proteins.

L4 ANSWER 7 OF 33 MEDLINE on STN
AN 1998115534 MEDLINE Full-text
DN PubMed ID: 9455999
TI Presence of nociceptin (orphanin FQ) receptors in rat retina: comparison with receptors in striatum.
AU Makman M H; Dvorkin B
CS Department of Biochemistry, Albert Einstein College of Medicine, Bronx, NY 10461, USA.
SO European journal of pharmacology, (1997 Nov 5) 338 (2) 171-6.
Journal code: 1254354. ISSN: 0014-2999.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 19980319
Last Updated on STN: 19980319
Entered Medline: 19980312
AB Nociceptin (orphanin FQ), a heptadecapeptide with some sequence homology to dynorphin A, has been proposed as an endogenous ligand for a previously cloned orphan receptor with significant homology to opioid receptors. Utilizing [(125)I][Tyr14]nociceptin as ligand, saturable and high affinity nociceptin binding sites were detected and characterized in rat retina and striatum. For retina, Bmax = 44.0 +/- 4.5 fmol/mg and Kd = 32.4 +/- 2.7 pM; for striatum, Bmax = 51.6 +/- 7.7 fmol/mg and Kd = 98.6 +/- 11.3 pM. In competition studies, nociceptin bound with picomolar affinity, dynorphin A with nanomolar affinity, naloxone and dynorphin A-(1-8) with micromolar affinity, while [des-Tyr1]dynorphin (dynorphin A-(2-17)), several other opioids, morphine and benzomorphans failed to compete for binding at 1-10 microM. Gpp(NH)p plus NaCl markedly decreased binding, consistent with involvement of a G protein-linked receptor. It is concluded that rat retina contains nociceptin receptors similar in concentration to those present in striatum. Properties of both the retinal and the striatal receptors are similar to those previously found for rat hypothalamus.

L4 ANSWER 8 OF 33 MEDLINE on STN
AN 1998021380 MEDLINE Full-text
DN PubMed ID: 9378852
TI Anatomical distribution of mu, delta, and kappa opioid- and nociceptin/orphanin FQ-stimulated [³⁵S]guanylyl-5'-O-(gamma-thio)-triphosphate binding in guinea pig brain.
AU Sim L J; Childers S R
CS Department of Physiology and Pharmacology, Center for the Neurobiological Investigation of Drug Abuse, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, North Carolina 27157, USA.
NC CA-12197 (NCI)
DA-00287 (NIDA)
DA-02904 (NIDA)

SO Journal of comparative neurology, (1997 Oct 6) 386 (4) 562-72.
Journal code: 0406041. ISSN: 0021-9967.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 20000303
Entered Medline: 19971110
AB An in vitro autoradiographic technique has recently been developed to visualize receptor-activated G-proteins by using agonist-stimulated [35S]guanylyl-5'-O-(gamma-thio)- triphosphate ([35S]GTPgammaS) binding in the presence of excess guanosine 5'-diphosphate. This technique was used to localize opioid-activated G-proteins in guinea pig brain, a species that contains the three major types of opioid receptors. This study used selective mu, delta, and kappa opioid agonists as well as nociceptin or orphanin FQ (N/OFQ) peptide, an endogenous ligand for an orphan opioid receptor-like (ORL1) receptor, to stimulate [35S]GTPgammaS binding in guinea pig brain sections. Opioid receptor specificity was confirmed by blocking agonist-stimulated [35S] GTPgammaS binding with the appropriate antagonists. In general, the distribution of agonist-stimulated [35S]GTPgammaS binding correlated with previous reports of receptor binding autoradiography, although quantitative differences suggest regional variations in receptor coupling efficiency. Mu, delta, and kappa opioid-stimulated [35S]GTPgammaS binding was found in the caudate-putamen, nucleus accumbens, amygdala, and hypothalamus. Mu-stimulated [35S]GTPgammaS binding predominated in the hypothalamus, amygdala, and brainstem, whereas kappa-stimulated [35S]GTPgammaS binding was particularly high in the substantia nigra and cortex and was moderate in the cerebellum. N/OFQ-stimulated [35S] GTPgammaS binding was highest in the cortex, hippocampus, and hypothalamus and exhibited a unique anatomical distribution compared with opioid-stimulated [35S]GTPgammaS binding. The present study extends previous reports on opioid and ORL1 receptor localization by anatomically demonstrating functional activity produced by mu, delta, and kappa opioid and ORL1 receptor activation of G-proteins.

L4 ANSWER 9 OF 33 MEDLINE on STN
AN 1998010570 MEDLINE Full-text
DN PubMed ID: 9346876
TI A beta-arrestin/green fluorescent protein biosensor for detecting G protein-coupled receptor activation.
AU Barak L S; Ferguson S S; Zhang J; Caron M G
CS Howard Hughes Medical Institute Laboratories and Department of Cell Biology, Duke University Medical Center, Durham, North Carolina 27710, USA.
NC HL 03422 (NHLBI)
NS 19576 (NINDS)
SO Journal of biological chemistry, (1997 Oct 31) 272 (44)
27497-500.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199712
ED Entered STN: 19980109
Last Updated on STN: 20000303
Entered Medline: 19971209
AB G protein-coupled receptors (GPCR)

represent the single most important drug targets for medical therapy, and information from genome sequencing and genomic data bases has substantially accelerated their discovery. The lack of a systematic approach either to identify the function of a new GPCR or to associate it with a cognate ligand has added to the growing number of orphan receptors. In this work we provide a novel approach to this problem using a beta-arrestin2/green fluorescent protein conjugate (betaarr2-GFP). It provides a real-time and single cell based assay to monitor GPCR activation and GPCR-G protein-coupled receptor kinase or GPCR-arrestin interactions. Confocal microscopy demonstrates the translocation of betaarr2-GFP to more than 15 different ligand-activated GPCRs. These data clearly support the common hypothesis that the beta-arrestin binding of an activated receptor is a convergent step of GPCR signaling, increase by 5-fold the number of GPCRs known to interact with beta-arrestins, demonstrate that the cytosol is the predominant reservoir of biologically active beta-arrestins, and provide the first direct demonstration of the critical importance of G protein-coupled receptor kinase phosphorylation to the biological regulation of beta-arrestin activity and GPCR signal transduction in living cells. The use of betaarr2-GFP as a biosensor to recognize the activation of pharmacologically distinct GPCRs should accelerate the identification of orphan receptors and permit the optical study of their signal transduction biology intractable to ordinary biochemical methods.

L4 ANSWER 10 OF 33 MEDLINE on STN
AN 97467951 MEDLINE Full-text
DN PubMed ID: 9324949
TI A bioluminescent assay for agonist activity at potentially any G-protein-coupled receptor.
AU Stables J; Green A; Marshall F; Fraser N; Knight E; Sautel M; Milligan G; Lee M; Rees S
CS Receptor Systems Unit, Glaxo Wellcome Research and Development, Stevenage, Herts, United Kingdom.
SO Analytical biochemistry, (1997 Oct 1) 252 (1) 115-26.
Journal code: 0370535. ISSN: 0003-2697.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 20021218
Entered Medline: 19971117
AB Transient expression of apoaequorin in Chinese hamster ovary (CHO) cells and reconstitution with the co-factor coelenterazine resulted in a large, concentration-dependent agonist-mediated luminescent response following cotransfection with the endothelin ETA, angiotensin ATII, thyrotropin-releasing hormone (TRH), and neurokinin NK1 receptors , all of which interact predominantly with the G alpha q-like phosphoinositidase-linked G-proteins. A substantially greater luminescence was obtained with mitochondrially targeted apoaequorin compared to cytoplasmically expressed apoaequorin. To generate a system amenable for the study of agonist activity at virtually any G-protein-coupled receptor the alpha subunit of the receptor promiscuous G-protein G alpha 16 was either transiently or stably expressed in CHO cells together with apoaequorin. In cells expressing G alpha 16, but not in its absence, agonists at a series of receptors which normally interact with either G alpha s or G alpha i were now able to cause a luminescent response from mitochondrially targeted apoaequorin. In the case of the A1 adenosine receptor, this response was clearly a result of activation of G alpha 16 and not a consequence of the release of the G alpha i-associated beta/gamma

complex, as the luminescent response was unaffected by pertussis toxin treatment of the cells, whereas agonist-mediated inhibition of adenylyl cyclase activity was attenuated. These studies describe the use of coexpressed apoaequorin as a reporter for G-protein-coupled receptor-mediated calcium signaling. Furthermore, coexpression of G alpha 16 and apoaequorin provides a basis for a generic mammalian cell microplate assay for the assessment of agonist action at virtually any G-protein-coupled receptor, including orphan receptors for which the physiological signal transduction mechanism may be unknown.

L4 ANSWER 11 OF 33 MEDLINE on STN
AN 97438287 MEDLINE Full-text
DN PubMed ID: 9292776
TI Molecular approaches to receptors as targets for drug discovery.
AU Herz J M; Thomsen W J; Yarbrough G G
CS Applied Receptor Sciences, Mill Creek, WA 98012, USA.
SO Journal of receptor and signal transduction research, (1997 Sep)
17 (5) 671-776. Ref: 275
Journal code: 9509432. ISSN: 1079-9893.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 20000303
Entered Medline: 19971104
AB The cloning of a great number of receptors and channels has revealed that many of these targets for drug discovery can be grouped into superfamilies based on sequence and structural similarities. This review presents an overview of how molecular biological approaches have revealed a plethora of receptor subtypes, led to new definitions of subtypes and isoforms, and played a role in the development of high selective drugs. Moreover, the diversity of subtypes has molded current views of the structure and function of receptor families. Practical difficulties and limitations inherent in the characterization of the ligand binding and signaling properties of expressed recombinant receptors are discussed. The importance of evaluating drug-receptor interactions that differ with temporally transient and distinct receptor conformational states is emphasized. Structural motifs and signal transduction features are presented for the following major receptor superfamilies: ligand-gated ion channel, voltage-dependent ion channel, G -protein coupled, receptor tyrosine-kinase, receptor protein tyrosine-phosphatase, cytokine and nuclear hormone. In addition, a prototypic receptor is analyzed to illustrate functional properties of a given family. The review concludes with a discussion of future directions in receptor research that will impact drug discovery, with a specific focus on orphan receptors as targets for drug discovery. Methods for classifying orphan receptors based upon homologies with members of existing superfamilies are presented together with molecular approaches to the greater challenge of defining their physiological roles. Besides revealing new orphan receptors, the human genome sequencing project will result in the identification of an abundance of novel receptors that will be molecular targets for the development of highly selective drugs. These findings will spur the discovery and development of an exciting new generation of receptor-subtype specific drugs with enhanced therapeutic specificity.

AN 97404870 MEDLINE Full-text
DN PubMed ID: 9261564
TI Molecular characterization and functional expression of opioid receptor-like1 receptor.
AU Wu Y L; Pu L; Ling K; Zhao J; Cheng Z J; Ma L; Pei G
CS Shanghai Institute of Cell Biology, Chinese Academy of Sciences, China.
SO Cell research, (1997 Jun) 7 (1) 69-77.
Journal code: 9425763. ISSN: 1001-0602.
CY China
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199709
ED Entered STN: 19971008
Last Updated on STN: 200000303
Entered Medline: 19970919
AB The opioid receptor-like1 receptor (ORL1), an orphan receptor whose human and murine complementary DNAs, has been characterized recently. ORL1 transcripts are particularly abundant in the central nervous system. We demonstrated that ORL1 expressed in human neuroblastoma SK-N-SH and SH-SY5Y cell lines by radioligand binding assay, reverse transcription polymerase chain reaction (RT-PCR) and Northern analysis in the present study. Stimulation with ORL1 specific agonist, nociceptin/orphanin FQ, increased [³⁵S]GTP gamma S binding to SK-N-SH cell membranes (EC₅₀ = 14 +/- 0.45 nM), and attenuated forskolin-stimulated accumulation of cellular cAMP (EC₅₀ = 0.80 +/- 0.45 nM), indicative that activation of ORL1 activates G proteins and inhibits adenylyl cyclase. Activation of ORL1 receptor was also accessed using CHO:hORL1 cell line by microphysiometer. Treatment of nociceptin/orphanin FQ increased extracellular acidification rate significantly.

L4 ANSWER 13 OF 33 MEDLINE on STN
AN 97382456 MEDLINE Full-text
DN PubMed ID: 9240460
TI The 6H1 orphan receptor, claimed to be the p2y5 receptor, does not mediate nucleotide-promoted second messenger responses.
AU Li Q; Schachter J B; Harden T K; Nicholas R A
CS Department of Pharmacology, University of North Carolina at Chapel Hill, 27599-7365, USA.
NC GM38213 (NIGMS)
SO Biochemical and biophysical research communications, (1997 Jul 18) 236 (2) 455-60.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199708
ED Entered STN: 19970908
Last Updated on STN: 19970908
Entered Medline: 19970828
AB An orphan G protein-coupled receptor , termed 6H1, with approximately 30% sequence identity to P2Y receptors has been proposed to be a P2Y receptor (p2y5) based solely on a radioligand binding assay with [³⁵S]-dATP alphaS [Webb et al. (1997) Biochem. Biophys. Res. Commun. 219:105-110]. Previous work in our laboratory has shown that [³⁵S]-dATP alphaS is not a general ligand for P2Y receptors, and thus inclusion of the p2y5 receptor in the family of P2Y receptors is questionable. To define unambiguously whether the p2y5 receptor is a P2Y receptor, we have cloned the turkey homologue of the chick p2y5 receptor. Sequence analysis indicated that the turkey receptor contains an

additional 32 amino acids at its carboxy terminus compared to the published chick sequence. HA epitope-tagged turkey p2y5 receptors were stably expressed in 1321N1 human astrocytoma cells, and cells shown to express the HA-tagged p2y5 receptor by an intact cell-based ELISA were used to determine whether changes in second messenger levels occurred in response to a series of nucleotides. ATP, ADP, UTP, UDP, dATP alphaS, and A2P4 had no effect on either inositol phosphate or cyclic AMP concentrations in cells expressing the p2y5 receptor. Robust inositol phosphate and cyclic AMP responses occurred to other G protein-coupled receptors expressed in 1321N1 cells, which indicate that these cells contain all of the necessary signaling components to generate these second messenger responses. These data indicate that the 6H1/p2y5 receptor is not a member of the P2Y receptor family of signaling proteins.

L4 ANSWER 14 OF 33 MEDLINE on STN
AN 97366605 MEDLINE Full-text
DN PubMed ID: 9223435
TI Cloning of a human heptahelical receptor closely related to the P2Y5 receptor.
AU Janssens R; Boeynaems J M; Godart M; Communi D
CS Institute of Interdisciplinary Research, School of Medicine, Erasme Hospital, Universite Libre de Bruxelles, Brussels, Belgium.
SO Biochemical and biophysical research communications, (1997 Jul 9) 236 (1) 106-12.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF005419
EM 199708
ED Entered STN: 19970813
Last Updated on STN: 19980206
Entered Medline: 19970807
AB The 6H1 receptor cloned from activated chicken T cells was initially considered an orphan G-coupled receptor, but was later included in the P2Y family of receptors for purine and pyrimidine nucleotides on the basis of a significant amino acid identity and was renamed P2Y5. Analysis of the expressed sequence tag database revealed the presence of a related sequence exhibiting 63% amino acid identity with this receptor. Starting from this partial sequence, we have isolated a complete clone and identified a 1113 base pair open reading frame encoding a new G-coupled receptor that we have called P2Y5-like. This sequence exhibits 61% identity with the chicken P2Y5 sequence and 30-33% with other P2Y subtypes. A construct encoding this P2Y5-like receptor was transfected into COS-7, 1321N1, and CHO-K1 cells, and expression was documented by Northern blotting. None of the 40 nucleotides and nucleosides tested was able to elicit a response in any of four functional assays: inositol phosphate formation, stimulation or inhibition of cAMP formation, and extracellular acidification measured with a microphysiometer. These results suggest either that the natural ligand of the P2Y5-like receptor is an uncommon nucleotide or alternatively that despite its structural similarity to the P2Y family it is not a nucleotide receptor.

L4 ANSWER 15 OF 33 MEDLINE on STN
AN 97312546 MEDLINE Full-text
DN PubMed ID: 9168987
TI Molecular cloning and tissue expression of a novel orphan G protein-coupled receptor from rat lung.
AU Bonini J A; Anderson S M; Steiner D F

CS Howard Hughes Medical Institute, University of Chicago, Illinois 60637,
USA.. jabonini@midway.uchicago.edu
SO Biochemical and biophysical research communications, (1997 May 8)
234 (1) 190-3.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U92802
EM 199706
ED Entered STN: 19970709
Last Updated on STN: 20000303
Entered Medline: 19970624
AB G protein-coupled receptors transduce the signal of a wide variety of hormones, neurotransmitters, cytokines, and other molecules across the cell membrane to elicit the corresponding response inside the target cells. We describe in this paper the molecular cloning and tissue distribution of a novel rat G protein-coupled receptor, GPR41, with highest homology to the human orphan G protein-coupled receptor DRY12. A lower degree of homology was seen with the receptors for bradykinin, angiotensin, and IL8. The expression of GPR41 appears to be the highest in brain and lung tissues, with lesser expression in heart, skeletal muscle, and kidney, as assayed by northern blotting. No GPR41 message was seen in spleen, liver, or testes. GPR41 failed to bind any of the ligands tested.

L4 ANSWER 16 OF 33 MEDLINE on STN
AN 97165887 MEDLINE Full-text
DN PubMed ID: 9013780
TI Cloning, characterization, and chromosomal localization of rec1.3, a member of the G-protein-coupled receptor family highly expressed in brain.
AU Macrae A D; Premont R T; Jaber M; Peterson A S; Lefkowitz R J
CS Department of Medicine (Cardiology), Howard Hughes Medical Institute, Durham, NC 27710, USA.
NC HL 16037 (NHLBI)
SO Brain research.. Molecular brain research, (1996 Dec) 42 (2) 245-54.
Journal code: 8908640. ISSN: 0169-328X.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U18405; GENBANK-U48235; GENBANK-U48236
EM 199704
ED Entered STN: 19970424
Last Updated on STN: 20000303
Entered Medline: 19970416
AB During a project to identify G-protein-coupled receptors (GPCR) expressed within taste buds, we have isolated a novel receptor-like sequence. The full length sequence of this receptor (rec1.3) has been obtained in both cow and mouse. Rec1.3 bears little sequence similarity to any GPCR whose ligand is known: the closest identity (33%) is to the orphan receptor edg-1. In cow, rec1.3 is expressed most prominently in the brain, with moderate expression in testis and tongue; in the mouse the expression is more widespread. No specific binding for a range of ligands was detected when the mouse coding sequence was expressed in eukaryotic cells. In situ hybridization showed that rec1.3 is widely expressed throughout the mouse brain and is highly expressed in localized regions of the hindbrain, midbrain and hypothalamus. The rec1.3

gene was localized to the centromeric region of chromosome 4 in mouse, a region associated with neonatal seizures.

L4 ANSWER 17 OF 33 MEDLINE on STN
AN 96430916 MEDLINE Full-text
DN PubMed ID: 8833998
TI Cloning and characterization of a new member of the G-protein coupled receptor EDG family.
AU Masana M I; Brown R C; Pu H; Gurney M E; Dubocovich M L
CS Department of Molecular Pharmacology and Biological Chemistry,
Northwestern University Medical School, Chicago, IL 60611, USA.
NC MH42922 (NIMH)
NS07140 (NINDS)
SO Receptors & channels, (1995) 3 (4) 255-62.
Journal code: 9315376. ISSN: 1060-6823.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U18405
EM 199611
ED Entered STN: 19961219
Last Updated on STN: 20000303
Entered Medline: 19961127
AB We report here the cloning of a new member of the endothelium differentiation gene (edg) subfamily of G-protein -coupled receptors. This novel cDNA sequence was cloned from the ovine pars tuberalis using a reverse transcriptase polymerase chain reaction (RT-PCR) amplification with degenerate primers homologous to the highly conserved II and VII transmembrane domains of the G- protein coupled receptor gene family. The PCR product was random primed with 32P and used as a probe to screen a size-selected cDNA ovine pars tuberalis library, which resulted in the isolation of a single clone of 2700 bp. This novel sequence was named edg-2, because its nucleic acid sequence was 55% homologous over 501nt overlap to an orphan sequence cloned from human endothelial cells, the endothelial differentiation gene, edg-1. The highest degree of aminoacid homology (42%) occurs in the seven putative transmembrane domains, particularly between the transmembrane domains III and VI (53% and 64%, respectively). The intervening hydrophilic domains are short and there are numerous putative phosphorylation sites for Ser/Thr-protein kinases in the second and third intracellular loop and in the COOH-terminal domain. Through Northern analysis of total RNA, low levels of at least four transcripts of 2.3, 2.5, 3.2 and 4 kb were found in sheep cerebral cortex and a 4.2 kb transcript was observed in NIH/3T3 fibroblasts. In addition, edg-2 transcripts (415 bp) were amplified by RT-PCR from pars tuberalis, cerebral blood vessels, hypothalamus, and retina. Serum stimulation of Chinese hamster ovary (CHO) cells expressing the edg-2 receptor resulted in increased cell proliferation, as measured by [3H]-thymidine incorporation. Edg-1 and edg-2 appear to be distinct genes that may encode protein products that bind the same or related ligand.

L4 ANSWER 18 OF 33 MEDLINE on STN
AN 96228068 MEDLINE Full-text
DN PubMed ID: 8647286
TI Cloning of a melatonin-related receptor from human pituitary.
AU Reppert S M; Weaver D R; Ebisawa T; Mahle C D; Kolakowski L F Jr
CS Laboratory of Developmental Chronobiology, Massachusetts General Hospital,
Boston, MA 02114,. USA.reppert@helix.mgh.harvard.edu
NC R37 HD14427 (NICHD)

SO FEBS letters, (1996 May 20) 386 (2-3) 219-24.
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U25341; GENBANK-U52218; GENBANK-U52219; GENBANK-U52220;
GENBANK-U52221
EM 199607
ED Entered STN: 19960805
Last Updated on STN: 20000303
Entered Medline: 19960725
AB We have cloned an orphan G protein-coupled receptor from a human pituitary cDNA library using a probe generated by PCR. The cDNA, designated H9, encodes a protein of 613 amino acids that is 45% identical at the amino acid level to the recently cloned human Mel(1a) and Mel(1b) melatonin receptors. Structural analyses of the encoded protein and its gene, along with phylogenetic analysis, further show that H9 is closely related to the G protein-coupled melatonin receptor family. Unusual features of the protein encoded by H9 include a lack of N-linked glycosylation sites and a carboxyl tail >300 amino acids long. H9 transiently expressed in COS-1 cells did not bind [¹²⁵I]melatonin or [³H]melatonin. H9 mRNA is expressed in hypothalamus and pituitary, suggesting that the encoded receptor and its natural ligand are involved in neuroendocrine function.

L4 ANSWER 19 OF 33 MEDLINE on STN
AN 96190677 MEDLINE Full-text
DN PubMed ID: 8619790
TI Identification of 6H1 as a P2Y purinoceptor: P2Y5.
AU Webb T E; Kaplan M G; Barnard E A
CS Molecular Neurobiology Unit, Royal Free Hospital School of Medicine,
London, United Kingdom.
SO Biochemical and biophysical research communications, (1996 Feb 6)
219 (1) 105-10.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-L06109
EM 199606
ED Entered STN: 19960620
Last Updated on STN: 19980206
Entered Medline: 19960613
AB We have determined the identity of the orphan G- protein coupled receptor cDNA, 6H1, present in activated chicken T cells, as a subtype of P2Y purinoceptor. This identification is based on first on the degree of sequence identity shared with recently cloned members of the P2Y receptor family and second on the pharmacological profile. Upon transient expression in COS-7 cells the 6H1 receptor bound the radiolabel [³⁵S]dATP alpha S specifically and with high affinity (K_d , 10 nM). This specific binding could be competitively displaced by a range of ligands active at P2 purinoceptors, with ATP being the most active (K_i), 116 nM). Such competition studies have established the following rank order of activity: ATP ADP 2-methylthioATP alpha, beta-methylene ATP, UTP, thus confirming 6H1 as a member of the growing family of P2Y purinoceptors. As the fifth receptor of this type to be identified we suggest that it be named P2Y5.

L4 ANSWER 20 OF 33 MEDLINE on STN
AN 96187229 MEDLINE Full-text
DN PubMed ID: 8777583
TI Leukotriene receptors.
AU Metters K M
CS Department of Biochemistry and Molecular Biology, Merck Frosst Centre for Therapeutic Research, Dorval, Quebec, Canada.
SO Journal of lipid mediators and cell signalling, (1995 Oct) 12 (2-3) 413-27. Ref: 126
Journal code: 9430888. ISSN: 0929-7855.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199609
ED Entered STN: 19960924
Last Updated on STN: 19970203
Entered Medline: 19960919
AB The current challenge in research on leukotriene receptors is to clone these molecules. Traditional protein purification approaches have not been successful in providing sequence information. Solubilization of cys-LT1 has been achieved but results in the dissociation of G-proteins and the loss of high affinity binding (Mong et al., 1986b; Mong and Sarau, 1990), while cys-LT2 activity cannot be monitored by other than functional assays and there have not been any purification attempts. Partial purification of B-LT has been reported but has not been continued to homogeneity (Sherman et al., 1992; Votta et al., 1990; Miki et al., 1990). Nor have attempts to clone these receptors through either homology screening or expression cloning been successful. The cloning of the prostanoid receptors, described in detail elsewhere in this volume, has shown that these receptors belong to a distinct family within the G-protein-coupled receptor superfamily. It is probable, therefore, that the leukotriene receptors will also belong to a separate group within this superfamily since phylogenetic comparisons have shown that receptors displaying high affinity for structurally related ligands exist as discrete families. Recently, a human cDNA encoding an orphan FMLP-related receptor cloned from HL60 cells of myeloid lineage was identified as the receptor for another eicosanoid, lipoxin A (Fiore et al., 1994). FMLP has a similar profile of biological actions to LTB4. Moreover, LTD4 showed a high degree of cross-reactivity with this receptor with an affinity only 20-fold less that of lipoxin A, although LTB4 was inactive. It remains to be determined whether the leukotriene receptors will fall into this class of receptors. The cloning of the leukotriene receptors will allow identification of the different receptor types and subtypes and potentially splice variants. Evaluation of currently developed antagonists at these receptor types could also open the way for novel therapies for inflammatory conditions.

L4 ANSWER 21 OF 33 MEDLINE on STN
AN 96055113 MEDLINE Full-text
DN PubMed ID: 7481766
TI Orphanin FQ: a neuropeptide that activates an opioidlike G protein-coupled receptor.
AU Reinscheid R K; Nothacker H P; Bourson A; Ardati A; Henningsen R A; Bunzow J R; Grandy D K; Langen H; Monsma F J Jr; Civelli O
CS Pharma Division, Hoffmann-La Roche AG, Basel, Switzerland.
NC DA 08562 (NIDA)
DA 09620 (NIDA)
SO Science, (1995 Nov 3) 270 (5237) 792-4.

CY Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199512
ED Entered STN: 19960124
Last Updated on STN: 20000303
Entered Medline: 19951206
AB A heptadecapeptide was identified and purified from porcine brain tissue as a ligand for an **orphan** heterotrimeric GTP- binding protein (G protein)-coupled receptor (LC132) that is similar in sequence to opioid receptors. This peptide, orphelin FQ, has a primary structure reminiscent of that of opioid peptides. Nanomolar concentrations of orphelin FQ inhibited forskolin-stimulated adenylyl cyclase activity in cells transfected with LC132. This inhibitory activity was not affected by the addition of opioid ligands, nor did the peptide activate opioid receptors. Orphelin FQ bound to its receptor in a saturable manner and with high affinity. When injected intracerebroventricularly into mice, orphelin FQ caused a decrease in locomotor activity but did not induce analgesia in the hot-plate test. However, the peptide produced hyperalgesia in the tail-flick assay . Thus, orphelin FQ may act as a transmitter in the brain by modulating nociceptive and locomotor behavior.

L4 ANSWER 22 OF 33 MEDLINE on STN
AN 96032699 MEDLINE Full-text
DN PubMed ID: 7559404
TI Identification of dynorphins as endogenous ligands for an opioid receptor-like **orphan** receptor.
AU Zhang S; Yu L
CS Department of Medical and Molecular Genetics, Indiana University School of Medicine, Indianapolis 46202, USA.
NC DA09116 (NIDA)
DA09444 (NIDA)
NS28190 (NINDS)
+
SO Journal of biological chemistry, (1995 Sep 29) 270 (39) 22772-6.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199511
ED Entered STN: 19951227
Last Updated on STN: 19970203
Entered Medline: 19951106
AB To identify the endogenous ligands for a cloned **orphan** receptor that shares high degrees of sequence homology with opioid receptors, this **orphan** receptor was expressed in Xenopus oocytes and in mammalian cell lines CHO-K1 and HEK-293. The coupling of the receptor to a G protein -activated K⁺ channel was used as a functional assay in oocytes. Endogenous opioid peptide dynorphins were found to activate the K⁺ channel by stimulating the **orphan** receptor. This activation was dose-dependent, with EC50 values at 45 and 37 nM for dynorphin A and dynorphin A-(1-13), respectively. The dynorphin effect was antagonized by the non-selective opioid antagonist naloxone but at rather high concentrations in the micromolar range. Naloxone also caused a rightward shift of the dose-response curve for dynorphin A, suggesting a competitive antagonism mechanism. In transiently transfected cells, 5 microM dynorphin A-(1-13) inhibited the forskolin-stimulated cyclic AMP increase by 51 and 35% in

CHO-K1 and HEK-293 cells, respectively. Other classes of endogenous opioids, i.e. enkephalins and endorphins, caused very little activation of this receptor. These results suggest that this **orphan receptor** is a member of the opioid receptor family and that dynorphins are endogenous ligands for this receptor.

L4 ANSWER 23 OF 33 MEDLINE on STN
AN 95374679 MEDLINE Full-text
DN PubMed ID: 7646814
TI Cloning, chromosomal localization, and RNA expression of a human beta chemokine receptor-like gene.
AU Combadiere C; Ahuja S K; Murphy P M
CS Laboratory of Host Defenses, National Institute of Allergy and Infectious Diseases, Bethesda, MD 20892, USA.
SO DNA and cell biology, (1995 Aug) 14 (8) 673-80.
Journal code: 9004522. ISSN: 1044-5498.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U28934
EM 199509
ED Entered STN: 19951005
Last Updated on STN: 19951005
Entered Medline: 19950925
AB A human cDNA encoding a putative G protein-coupled receptor designated chemokine beta receptor-like 1 (CMKBRL1) was isolated from an eosinophilic leukemia library. Its deduced sequence is approximately 40% identical to previously cloned receptors for the beta chemokines macrophage inflammatory protein-1 alpha (MIP-1 alpha), RANTES, and monocyte chemoattractant protein-1 (MCP-1), which are chemoattractants for blood leukocytes, and is 83% identical to the product of the orphan rat cDNA RBS 11. Like the MIP-1 alpha/RANTES receptor, CMK-BRL1 is encoded by a small, single-copy gene that maps to chromosome 3p21 and is expressed in leukocytes. However, two screening assays with a broad panel of chemokines failed to identify its ligand. CMKBRL1 mRNA was detectable by Northern blot hybridization in neutrophils and monocytes, but not eosinophils, and was also found in eight solid organs that were tested with particularly high expression in brain. The RNA distribution of the known beta chemokine receptors was overlapping but distinct from that of CMKBRL1. MIP-1 alpha/RANTES receptor mRNA was detectable in neutrophils, monocytes, eosinophils, and in all eight solid organs tested, with particularly high expression in placenta, lung, and liver. MCP-1 receptor mRNA was found in monocytes, lung, liver, and pancreas. These results suggest that the ligand for the putative CMKBRL1 receptor is a beta chemokine that targets both neutrophils and monocytes. Moreover, the RNA distributions suggest that CMKBRL1, the MIP-1 alpha/RANTES receptor, and the MCP-1 receptor may have both overlapping and distinct biological roles.

L4 ANSWER 24 OF 33 MEDLINE on STN
AN 95366960 MEDLINE Full-text
DN PubMed ID: 7639700
TI Molecular cloning of an orphan G-protein
-coupled receptor that constitutively activates adenylylate cyclase.
AU Eggerickx D; Denef J F; Labbe O; Hayashi Y; Refetoff S; Vassart G;
Parmentier M; Libert F
CS Institut de Recherche Interdisciplinaire, Faculte de Medecine, Universite Libre de Bruxelles, Belgium.
NC DK 15070 (NIDDK)

SO Biochemical journal, (1995 Aug 1) 309 (Pt 3) 837-43.
Journal code: 2984726R. ISSN: 0264-6021.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X83956
EM 199509
ED Entered STN: 19950921
Last Updated on STN: 20020613
Entered Medline: 19950913
AB A human gene encoding an **orphan G-protein -coupled receptor** named ACCA (adenylate cyclase constitutive activator) was isolated from a genomic library using as a probe a DNA fragment obtained by low-stringency PCR. Human ACCA (hACCA) is a protein of 330 amino acids that exhibits all the structural hallmarks of the main family of G-protein-coupled receptors. Expression of hACCA resulted in a dramatic stimulation of adenylate cyclase, similar in amplitude to that obtained with other Gs-coupled receptors fully activated by their respective ligands. This stimulation was obtained in a large variety of stable cell lines derived from various organs, and originating from different mammalian species. hACCA was found to be the human homologue of a recently reported mouse **orphan receptor** (GPCR21). The mouse ACCA (mACCA) was therefore recloned by PCR, and expression of mACCA in Cos-7 cells demonstrated that the mouse receptor behaved similarly as a constitutive activator of adenylate cyclase. It is not known presently whether the stimulation of adenylate cyclase is the result of a true constitutive activity of the receptor or, alternatively, is the consequence of a permanent stimulation by a ubiquitous ligand. The tissue distribution of mACCA was determined by RNase protection assay. Abundant transcripts were found in the brain, whereas lower amounts were detected in testis, ovary and eye. Various hypotheses concerning the constitutive activity of ACCA and their potential biological significance are discussed.

L4 ANSWER 25 OF 33 MEDLINE on STN
AN 95171461 MEDLINE Full-text
DN PubMed ID: 7867073
TI The anticoagulation factor protein S and its relative, Gas6, are ligands for the Tyro 3/Axl family of receptor tyrosine kinases.
AU Stitt T N; Conn G; Gore M; Lai C; Bruno J; Radziejewski C; Mattsson K; Fisher J; Gies D R; Jones P F; +
CS Regeneron Pharmaceuticals, Incorporated, Tarrytown, New York 10591.
SO Cell, (1995 Feb 24) 80 (4) 661-70.
Journal code: 0413066. ISSN: 0092-8674.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199503
ED Entered STN: 19950407
Last Updated on STN: 20000303
Entered Medline: 19950328
AB We report the identification of ligands for Tyro 3 (alternatively called Sky, rse, brt, or tif) and Axl (alternatively, Ark or UFO), members of a previously orphan family of receptor-like tyrosine kinases. These ligands correspond to protein S, a protease regulator that is a potent anticoagulant, and Gas6, a protein related to protein S but lacking any known function. Our results are reminiscent of recent findings that the procoagulant thrombin, a protease that drives clot formation by cleaving fibrinogen to form fibrin, also binds and activates intracellular signaling via a G protein-coupled cell surface

receptor. Proteases and protease regulators that also activate specific cell surface receptors may serve to integrate coagulation with associated cellular responses required for tissue repair and growth, as well as to coordinate protease cascades and associated cellular responses in other systems, such as those involved in growth and remodeling of the nervous system.

L4 ANSWER 26 OF 33 MEDLINE on STN
AN 91266895 MEDLINE Full-text
DN PubMed ID: 1646713
TI The orphan receptor cDNA RDC7 encodes an A1 adenosine receptor.
AU Libert F; Schiffmann S N; Lefort A; Parmentier M; Gerard C; Dumont J E; Vanderhaeghen J J; Vassart G
CS Institut de Recherche Interdisciplinaire, Faculte de Medecine, Universite Libre de Bruxelles, Belgium.
SO EMBO journal, (1991 Jul) 10 (7) 1677-82.
Journal code: 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199107
ED Entered STN: 19910811
Last Updated on STN: 20000303
Entered Medline: 19910724
AB The extensive amino acid sequence conservation among G protein-coupled receptors has been exploited to clone new members of this large family by homology screening or by PCR. Out of four such receptor cDNAs we cloned recently, RDC7 corresponds to a relatively abundant transcript in the brain cortex, the thyroid follicular cell and the testis. We have now identified RDC7 as an A1 adenosine receptor. The A1 agonist CPA [N6-cyclopentyladenosine] decreased by 80% cAMP accumulation in forskolin-stimulated CHO cells stably transfected with RDC7. Specific binding of another A1 adenosine agonist, [³H]CHA [N6-cyclohexyladenosine], was demonstrated on membranes from Cos cells transfected with a pSVL construct harbouring the RDC7 cDNA insert. The binding characteristics were similar to those of the natural brain A1 receptor. The recombinant and the natural receptors behaved also in the same way in displacement experiments involving a series of A1 adenosine agonists. The binding characteristics of RDC7 were compared to those of RDC8, another orphan receptor recently identified as an A2 adenosine receptor. The two molecular species RDC7 and RDC8 correspond clearly to the A1 and A2 receptor entities defined hitherto on a purely pharmacological basis.

L4 ANSWER 27 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.
on STN
AN 1998:299739 SCISEARCH Full-text
GA The Genuine Article (R) Number: ZG655
TI New aspects of G-protein-coupled receptor signalling
and regulation
AU Milligan G (Reprint)
CS UNIV GLASGOW, INST BIOMED & LIFE SCI, DIV BIOCHEM & MOL BIOL, MOL PHARMACOL GRP, GLASGOW G12 8QQ, LANARK, SCOTLAND (Reprint)
CYA SCOTLAND
SO TRENDS IN ENDOCRINOLOGY AND METABOLISM, (JAN-FEB 1998) Vol. 9,
No. 1, pp. 13-19.
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010.
ISSN: 1043-2760.

DT General Review; Journal
FS LIFE
LA English
REC Reference Count: 52
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Research on the structure, regulation and signalling properties of the family of seven-transmembrane-helix, heterotrimeric guanine nucleotide-binding protein (G-protein)-coupled receptors (GPCRs) continues at a frantic pace. This reflects their central role in transmission of hormone- and neurotransmitter-encoded information across the plasma membrane of cells. The location of the ligand-binding sites on the extracellular face of the membrane has made them obvious targets for therapeutic intervention in a wide range of conditions resulting from endocrine imbalance. Furthermore, based on the identification of many novel GPCR sequences emerging from expressed sequence tags (ESTs) and other DNA sequencing programmes, it has become clear that the GPCR family is likely to be considerably larger than appreciated in even the recent past. Although neither the natural ligands nor synthetic pharmaceuticals have yet been identified for these so-called 'orphan' GPCRs, they offer the potential for a plethora of new therapeutic targets. Within a short review, it is impossible to cover all the current developments in this field and the topics selected represent a personal view of recent high-lights of areas that provide both novel and general insights into the function and regulation of GPCRs.

L4 ANSWER 28 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.
on STN

AN 1998:240418 SCISEARCH Full-text

GA The Genuine Article (R) Number: ZC567

TI A human gene encodes a putative G protein-coupled receptor highly expressed in the central nervous system

AU Donohue P J; Shapira H; Mantey S A; Hampton L L; Jensen R T; Battey J F (Reprint)

CS NATL INST DEAFNESS & OTHER COMMUN DISORDERS, MOL BIOL LAB, NIH, ROCKVILLE, MD 20850 (Reprint); NATL INST DEAFNESS & OTHER COMMUN DISORDERS, MOL BIOL LAB, NIH, ROCKVILLE, MD 20850; TEL AVIV UNIV, SACKLER FAC MED, DEPT PHYSIOL & PHARMACOL, IL-69978 RAMAT AVIV, ISRAEL; NIDDKD, DIGEST DIS BRANCH, NIH, BETHESDA, MD 20892

CYA USA; ISRAEL

SO MOLECULAR BRAIN RESEARCH, (FEB 1998) Vol. 54, No. 1, pp. 152-160.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0169-328X.

DT Article; Journal
FS LIFE
LA English
REC Reference Count: 16
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The mammalian bombesin (Bn)-like neuropeptide receptors gastrin-releasing peptide receptor (GRP-R) and neuromedin B receptor (NMB-R) transduce a variety of physiological signals that regulate secretion, growth, muscle contraction, chemotaxis and neuromodulation. We have used reverse transcription-polymerase chain reaction (PCR) to isolate a cDNA from human brain mRNA, GPCR /CNS, that encodes a putative G protein-coupled receptor (GPCR) based upon the presence of the paradigmatic seven heptahelical transmembrane domains in its predicted amino acid sequence. Analysis of the deduced protein sequence of GPCR/CNS reveals this putative receptor to be 98% identical to the deduced amino acid sequence of a recently reported

gene product and minimally identical (similar to 23%) to both murine GRP-R and human endothelin-B (ET-B) receptor. Our deduced protein sequence differs at 12 positions, scattered throughout the open reading frame, relative to the original sequence. A 3.7 kb GPCR/CNS mRNA species is expressed in vivo in a tissue-specific manner, with highest levels detected in brain and spinal cord, lower levels found in testis, placenta and liver, but no detectable expression observed in any other tissue. Analysis of GPCR/CNS genomic clones reveals that the human gene contains one intron that is about 21 kb in length that divides the coding region into two exons and maps to human chromosome 7q31. No specific binding is observed with either a newly identified ligand (DTyr(6),beta Ala(11),Phe(13),Nle(14)]Bn-(6-14)) having high affinity for all Bn receptor subtypes or Bn after GPCR/CNS is stably expressed in fibroblasts. No elevation in inositol trisphosphate is observed after the application of micromolar levels of either DPhe(6),beta Ala(11),Phe(13),Nle(14)]Bn-(6-14) or Bn, a concentration of agonist known to activate all four known Bn receptor subtypes. When GPCR/CNS is expressed in Xenopus oocytes, no activation of the calcium-dependent chloride channel is detected despite the addition of micromolar levels of Bn peptide agonists. We conclude that the natural ligand for this receptor is none of the known naturally occurring Bn-like peptides and the true agonist for GPCR/CNS remains to be elucidated. (C) 1998 Elsevier Science B.V.

L4 ANSWER 29 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.
on STN
AN 95:810225 SCISEARCH Full-text
GA The Genuine Article (R) Number: TF351
TI CLONING, SEQUENCING AND TISSUE DISTRIBUTION OF 2 RELATED G-
PROTEIN-COUPLED RECEPTOR CANDIDATES EXPRESSED PROMINENTLY IN HUMAN
LUNG-TISSUE
AU AN S Z (Reprint); TSAI C; GOETZL E J
CS UNIV CALIF SAN FRANCISCO, DEPT MED, DIV ALLERGY & IMMUNOL, SAN FRANCISCO,
CA, 94143 (Reprint); UNIV CALIF SAN FRANCISCO, DEPT IMMUNOL MICROBIOL, SAN
FRANCISCO, CA, 94143
CYA USA
SO FEBS LETTERS, (13 NOV 1995) Vol. 375, No. 1-2, pp. 121-124.
ISSN: 0014-5793.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 22
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB A novel G protein-coupled receptor, named GPR12A, was cloned by a PCR strategy using degenerate primers designed from sequences conserved among receptors for inflammatory mediators. Screening of a human lung cDNA library with GPR12A as a probe also identified a closely-related cDNA (GPR6C.1) that has been previously reported as GPR4 [13]. GPR12A and GPR6C.1 are 46.1% identical in amino acid sequence, but are less than 33% identical to any other known receptors. Northern analysis revealed that they are expressed prominently in the lung. Although the ligands for GPR12A and GPR6C.1 are unknown, their similarity suggests that they are receptors for ligands of similar or identical chemical nature.

L4 ANSWER 30 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.
on STN
AN 95:161028 SCISEARCH Full-text
GA The Genuine Article (R) Number: QH537
TI G-PROTEIN-COUPLED ORPHAN RECEPTORS

AU PARMENTIER M (Reprint); LIBERT F; VASSART G
CS FREE UNIV BRUSSELS, IRIBHN, FONDS NATL RECH SCI, CAMPUS ERASME, 808 ROUTE LENNIK, B-1070 BRUSSELS, BELGIUM (Reprint)
CYA BELGIUM
SO M S-MEDECINE SCIENCES, (FEB 1995) Vol. 11, No. 2, pp. 222-231.
ISSN: 0767-0974.
DT General Review; Journal
FS LIFE
LA French
REC No References Keyed
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB G protein-coupled receptors are encoded by one of the mammalian largest gene families. These receptors share a common transmembrane organization, respond to a large variety of structurally different ligands, and regulate intracellular enzymes and channels through heterotrimeric G proteins. The first genes encoding receptors of this family were isolated following the purification of the protein and sequencing of peptides generated by proteolytic cleavage. Subsequently, a number of G protein-coupled receptor genes were cloned through expression screening procedures based either on the binding of a specific ligand, or on the functional coupling of the recombinant receptor to a transduction cascade. Most members in this gene family were however obtained by homology cloning, using cross-hybridization or low-stringency polymerase chain reaction. The result is the availability of more than 140 receptor types and subtypes (olfactory receptors excluded). With a few exceptions, all pharmacologically well-defined receptors have now been cloned. Molecular cloning confirmed the existence of poorly characterized subtypes and uncovered other unsuspected subtypes. Genes encoding uncharacterized receptors have also been made available, either related to identified subfamilies, or defining new subfamilies by themselves. These so-called << orphan >> receptors are waiting for the identification of the corresponding ligands and of their biological function. The availability of cloned human receptors is expected to speed up the search for more specific drugs. Determination of the three dimensional structure of selected recombinant receptors, and computer modelling of receptor-ligand interaction and ligand-mediated receptor activation will probably allow in the long run the rational design of specific agonists and antagonists. The increasing number of reported orphan receptors will certainly be instrumental in the discovery of new regulatory pathways linking cells, with the potential of opening new avenues in pharmacology.

L4 ANSWER 31 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation.
on STN
AN 94:196245 SCISEARCH Full-text
GA The Genuine Article (R) Number: NC117
TI CDNA CLONING OF A G-PROTEIN-COUPLED RECEPTOR EXPRESSED
IN RAT SPINAL-CORD AND BRAIN RELATED TO CHEMOKINE RECEPTORS
AU HARRISON J K (Reprint); BARBER C M; LYNCH K R
CS UNIV FLORIDA, COLL MED, DEPT PHARMACOL & THERAPEUT, BOX 100267,
GAINESVILLE, FL, 32610 (Reprint); UNIV VIRGINIA, SCH MED, DEPT PHARMACOL,
CHARLOTTESVILLE, VA, 22908; UNIV VIRGINIA, SCH MED, DEPT BIOCHEM,
CHARLOTTESVILLE, VA, 22908
CYA USA
SO NEUROSCIENCE LETTERS, (14 MAR 1994) Vol. 169, No. 1-2, pp.
85-89.
ISSN: 0304-3940.
DT Article; Journal
FS LIFE
LA ENGLISH

REC Reference Count: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A series of cDNAs and a genomic clone (named RBS11) were isolated, from a variety of rat brainstem, pituitary and/or spinal cord cDNA libraries and a genomic library by low-stringency hybridization screening with a rat angiotensin receptor cDNA. The RBS11 protein, as conceptualized from these DNAs, is a novel member of the rhodopsin family of the G-protein-coupled receptor (GCR) superfamily. Comparison of RBS11 to other members of the GCR superfamily suggests that the RBS11 protein might be a receptor for a peptide ligand in the chemokine family. The RBS11 protein sequence is unusual in that it is without N-linked glycosylation consensus sequences in the putative exofacial regions. Northern analysis indicates that the mRNA for RBS11 accumulates widely and unevenly in the adult rat, with the mRNA being most prominent in extracts of spinal cord, brain, kidney, gut, uterus and testes.

L4 ANSWER 32 OF 33 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

AN 1997:207816 BIOSIS Full-text

DN PREV199799507019

TI Functional coupling of the nociceptin/orphanin FQ receptor with the G-protein-activated K⁺ (GIRK) channel.

AU Ikeda, Kazutaka [Reprint author]; Kobayashi, Katsunori; Kobayashi, Toru; Ichikawa, Tomio; Kumanishi, Toshiro; Kishida, Haruo; Yano, Ryoji; Manabe, Toshiya

CS Lab. Cell. Inf. Processing, Frontier Res. Program, Inst. Phys. Chem. Res., 2-1 Hirosawa, Wako, Saitama 351-01, Japan

SO Molecular Brain Research, (1997) Vol. 45, No. 1, pp. 117-126.
CODEN: MBREE4. ISSN: 0169-328X.

DT Article

LA English

ED Entered STN: 12 May 1997
Last Updated on STN: 2 Jun 1997

AB Nociceptin/orphanin FQ is a heptadecapeptide which was recently isolated from brains. It induces hyperalgesia, in contrast to the analgesic effects of opioid ligands, although it and its receptor structurally resemble opioid peptides and opioid receptors, respectively. To investigate the molecular mechanism underlying nociceptin/orphanin FQ actions, we performed Xenopus oocyte expression assays, in situ hybridization histochemistry and electrophysiological analyses of neurons. We found that the nociceptin/orphanin FQ receptor is functionally coupled with the G-protein-activated K⁺ (GIRK) channel in Xenopus oocytes, and that the receptor mRNA and GIRK1 mRNA co-exist in various neurons, including hippocampal pyramidal cells. Furthermore, we found that nociceptin/orphanin FQ induces hyperpolarizing currents via inward-rectifier K⁺ channels in hippocampal pyramidal cells, suggesting that the nociceptin/orphanin FQ receptor couples with the GIRK channel in this region. We conclude that the nociceptin/orphanin FQ receptor couples with the GIRK channel in various neurons, including hippocampal pyramidal cells, thereby modulating neuronal excitability.

L4 ANSWER 33 OF 33 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

AN 1995:381269 BIOSIS Full-text

DN PREV199598395569

TI Studies of ligand binding and signal transduction pathway of the chimeric receptor of IL-8 receptor and LESTR.

AU Na, S.; Ogborne, K. T.; Hinkel, C. A.; Neote, K. S.; Hanke, J. H.

CS Central Res., Pfizer Inc., Groton, CT, USA
SO 9TH INTERNATIONAL CONGRESS OF IMMUNOLOGY. (1995) pp. 111. The 9th International Congress of Immunology.
Publisher: 9th International Congress of Immunology, San Francisco, California, USA.
Meeting Info.: Meeting Sponsored by the American Association of Immunologists and the International Union of Immunological Societies. San Francisco, California, USA. July 23-29, 1995.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 1 Sep 1995
Last Updated on STN: 1 Sep 1995

=> FIL STNGUIDE

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|----------------------|------------------|---------------|
| FULL ESTIMATED COST | 51.43 | 51.64 |

FILE 'STNGUIDE' ENTERED AT 12:07:17 ON 15 JAN 2005
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2005 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jan 7, 2005 (20050107/UP).

=> d his

(FILE 'HOME' ENTERED AT 11:59:37 ON 15 JAN 2005)

FILE 'MEDLINE, SCISEARCH, EMBASE, BIOSIS' ENTERED AT 11:59:56 ON 15 JAN 2005

L1 2546 S RECEPTOR (P) (LIGAND OR AGONIST OR ANTAGONIST OR COMPOUND) (P
L2 912 S L1 AND PY<=1998
L3 284 DUP REM L2 (628 DUPLICATES REMOVED)
L4 33 S L3 AND (GPCR OR G (W) PROTEIN)

FILE 'STNGUIDE' ENTERED AT 12:07:17 ON 15 JAN 2005

=>

---Logging off of STN---

| Ref # | Hits | Search Query | DBs | Default Operator | Plurals | Time Stamp |
|-------|------|---|---|------------------|---------|------------------|
| L1 | 157 | (hinuma-shuji or hosoya-masaki).in. | US-PGPUB;
USPAT;
EPO; JPO;
DERWENT | OR | OFF | 2005/01/15 16:09 |
| L2 | 16 | I1 and orphan adj receptor | US-PGPUB;
USPAT;
EPO; JPO;
DERWENT | OR | OFF | 2005/01/15 16:12 |
| L3 | 379 | (orphan adj receptor) same
(LIGAND OR AGONIST OR
ANTAGONIST OR COMPOUND\$)
same (SCREEN\$ OR BIND\$ OR
ASSAY\$) | US-PGPUB;
USPAT;
EPO; JPO;
DERWENT | OR | OFF | 2005/01/15 16:13 |
| L4 | 42 | I3 and @py<="1999" | US-PGPUB;
USPAT;
EPO; JPO;
DERWENT | OR | OFF | 2005/01/15 16:14 |
| L5 | 58 | I2 or I4 | US-PGPUB;
USPAT;
EPO; JPO;
DERWENT | OR | OFF | 2005/01/15 16:14 |

East Search**15 January 2005**

| | | | |
|--------------------------|----------|-----------|---|
| US 20040254348 A1 | US-PGPUB | 20041216 | Novel polypeptide and dna thereof |
| US 20040248790 A1 | US-PGPUB | 20041209 | Novel secretory proteins and dna thereof |
| US 20040171067 A1 | US-PGPUB | 20040902 | Screening method |
| US 20040152136 A1 | US-PGPUB | 20040805 | Screening method |
| US 20040132073 A1 | US-PGPUB | 20040708 | Novel G protein-coupled receptor protein, its DNA and ligand thereof |
| US 20030092618 A1 | US-PGPUB | 20030515 | Polypeptides, their production and use |
| US 20030082648 A1 | US-PGPUB | 20030501 | Novel mass receptor-analogous protein and dnas thereof |
| US 20020168720 A1 | US-PGPUB | 20021114 | G protein coupled receptor proteins, their production and use |
| US 20020143152 A1 | US-PGPUB | 20021003 | Polypeptides, their production and use |
| US 20020009771 A1 | US-PGPUB | 20020124 | G PROTEIN COUPLED RECEPTOR PROTEINS, THEIR
PRODUCTION AND USE |
| US 6794491 B1 | USPAT | 20040921 | Polypeptides, their production and use |
| US 6492324 B1 | USPAT | 20021210 | APJ ligand polypeptides |
| US 6228984 B1 | USPAT | 20010508 | Polypeptides their production and use |
| US 6005086 A
same | USPAT | 199911221 | Farnesoid activated receptor polypeptides, and nucleic acid encoding the
same |
| US 5989834 A | USPAT | 19991123 | Uses of nucleic acid encoding neuropeptide Y/peptide YY (Y2) receptors |
| US 5989537 A | USPAT | 19991123 | Methods for stimulating granulocyte/macrophage lineage using |
| thrombopoietin | | | |
| US 5976834 A
receptor | USPAT | 19991102 | cDNA clone HNFJD15 that encodes a novel human 7-transmembrane
receptor |
| US 5958683 A | USPAT | 19990928 | Screening method using the RZR receptor family |
| US 5955303 A | USPAT | 19990921 | Human chemokine receptor-like protein |
| US 5945279 A | USPAT | 19990831 | Screening system for identifying compounds that regulate steroid and
orphan receptors mediation of |
| US 5942405 A | USPAT | 19990824 | DNA transcription |
| US 5912335 A | USPAT | 19990615 | Therapeutic and screening methods using C3a receptor and C3a
G-protein coupled receptor HUVCT36 |

| | | | | |
|--------------|-------|----------|--|--|
| US 5871916 A | USPAT | 19990216 | ECDN protein and DNA encoding the same | |
| US 5866346 A | USPAT | 19990202 | Methods of using dynorphins as ligands for XOR1 receptor | |
| US 5849485 A | USPAT | 19981215 | Liver enriched transcription factor | |
| US 5834213 A | USPAT | 19981110 | Screening system and assay for identifying compounds that regulate steroid and orphan receptors | |
| | | | mediation of DNA transcription | |
| US 5824504 A | USPAT | 19981020 | Human 7-transmembrane receptor and DNA | |
| US 5814479 A | USPAT | 19980929 | Bsk receptor-like tyrosine kinase | |
| US 5789170 A | USPAT | 19980804 | Specific co-activator for human androgen receptor | |
| US 5783402 A | USPAT | 19980721 | Method of identifying ligands and antagonists of G-protein coupled receptor | |
| US 5747661 A | USPAT | 19980505 | Retinoid-inducible response elements | |
| US 5714595 A | USPAT | 19980203 | Mechanism-based screen for retinoid X receptor agonists and antagonists | |
| US 5707800 A | USPAT | 19980113 | Retinoic acid response elements and assays employing same | |
| US 5705349 A | USPAT | 19980106 | Methods for preparing polynucleotides encoding orphan receptor ligands | |
| US 5700682 A | USPAT | 19971223 | Mechanism based screen for retinoid X receptor agonists and antagonists | |
| US 5700650 A | USPAT | 19971223 | Mechanism-based screen for retinoid X receptor agonists and antagonists | |
| US 5691196 A | USPAT | 19971125 | Recombinant nucleic acid containing a response element | |
| US 5639616 A | USPAT | 19970617 | Isolated nucleic acid encoding a ubiquitous nuclear receptor | |
| US 5604115 A | USPAT | 19970218 | Liver enriched transcription factor | |
| US 5597693 A | USPAT | 19970128 | Hormone response element compositions and assay | |
| US 5545549 A | USPAT | 19960813 | DNA encoding a human neuropeptide Y/neuropeptide YY (Y2) receptor and uses thereof | |
| US 5541085 A | USPAT | 19960730 | Method for preparing orphan receptor ligands | |
| US 5538861 A | USPAT | 19960723 | DNA encoding a stimulating factor for the axl receptor | |
| US 5532157 A | USPAT | 19960702 | Host cell line LVIP2.0Zc, useful in assays to identify ligands and ant agonists of G protein-coupled receptors | |
| US 5514578 A | USPAT | 19960507 | Polynucleotides encoding insect steroid hormone receptor polypeptides and cells transformed with same | |

| | | | |
|---|---------|---|--|
| JP 2004180668 A | JPO | 20040702 | METHOD FOR DETERMINING LIGAND |
| JP 2001128695 A | JPO | 20010515 | SCREENING METHOD |
| JP 411315099 A | JPO | 19991116 | PRODUCTION OF VARIANT TYPE ORPHAN RECEPTOR AND VARIANT TYPE |
| JP 10068724 A | JPO | 19980310 | NOCICEPTIN RECEPTOR |
| WO 3071272 A1 | EPO | 20030828 | SCREENING METHOD FOR LC132 (OPIOID) RECEPTOR AGONIST |
| JP 11315099 A | DERWENT | 19991116 | METHOD OF DETERMINING LIGAND |
| A process for preparation of a variant orphan receptor and a variant nociceptin receptor - useful for | | A process for preparation of a variant orphan receptor and a variant nociceptin receptor - useful for | |
| US 20020143152 A | DERWENT | 20021003 | the development of opioid analgesics with reduced adverse reactions |
| treating disorders of | | New polypeptide ligand for orphan G protein coupled receptors - used for | |
| US 5597693 A | DERWENT | 19970128 | central nervous system, pituitary and pancreas, and for drug screening |
| determining target gene | | Steroid/thyroid hormone receptor DNA-binding domain compsns. - for | |
| receptors and for altering target | | specificity, also methods for identifying ligands that activate orphan | |
| US 6458926 B | DERWENT | 20021001 | gene specificity |
| useful in transduction of | | Heterodimeric complex of retinoid X receptor (RXR) and a silent partner - | |
| US 5541085 A | DERWENT | 19960730 | novel responses to multiple hormonal signals |
| which express the orphan | | Isolation of orphan receptor ligands - by mutagenising transfected cells | |
| WO 9313129 A | DERWENT | 19930708 | receptor and obtaining ligands from surviving cells |
| enhances or inhibits | | Hetero-dimer comprising retinoid X receptor and hormone receptor - | |
| contg. hormone receptor | | transcription, used for screening ligands for effect on activity or RXR- | |
| US 5707800 A | DERWENT | 19980113 | hetero-dimers |
| expression in cells and for | | Retinoic acid response element - used for controlling recombinant DNA | |

EP 463081 B
for identifying ligands for

DERWENT 19980520

screening agonists and antagonists
Steroid and thyroid hormone response elements - used to develop assays
orphan hormone receptors

Day : Saturday
Date: 1/15/2005

Time: 16:07:59

PALM INTRANET

Inventor Information for 10/069228

| Inventor Name | City | State/Country |
|----------------|---------|---------------|
| HINUMA, SHUJI | IBARAKI | JAPAN |
| HOSOYA, MASAKI | IBARAKI | JAPAN |

[Appln Info](#) [Contents](#) [Petition Info](#) [Atty/Agent Info](#) [Continuity Data](#) [Foreign Data](#)

Search Another: Application# or Patent#
PCT / / or PG PUBS #
Attorney Docket #
Bar Code #

To go back use Back button on your browser toolbar.

Back to [PALM](#) | [ASSIGNMENT](#) | [OASIS](#) | Home page

Day : Saturday
 Date: 1/15/2005

Time: 16:08:01


PALM INTRANET
Inventor Name Search Result

Your Search was:

Last Name = HINUMA

First Name = SHUJI

| Application# | Patent# | Status | Date Filed | Title | Inventor Name 51 |
|---------------------------------|----------------|---------------|-------------------|--|-------------------------|
| <u>60017954</u> | Not Issued | 159 | 05/20/1996 | NOVEL HUMAN G-PROTEIN COUPLED RECEPTOR | HINUMA , SHUJI |
| <u>60017915</u> | Not Issued | 159 | 05/16/1996 | NOVEL HUMAN G-PROTEIN COUPLED RECEPTOR (HIBCD07) | HINUMA , SHUJI |
| <u>10914735</u> | Not Issued | 019 | 08/09/2004 | G-PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES | HINUMA, SHUJI |
| <u>10719587</u> | Not Issued | 020 | 11/21/2003 | NOVEL G PROTEIN-COUPLED RECEPTOR PROTEIN, ITS DNA AND LIGAND THEREOF | HINUMA, SHUJI |
| <u>10706703</u> | Not Issued | 160 | 11/12/2003 | G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES | HINUMA, SHUJI |
| <u>10505486</u> | Not Issued | 019 | 01/01/0001 | METHOD OF DETERMINING LIGAND | HINUMA, SHUJI |
| <u>10504726</u> | Not Issued | 020 | 08/13/2004 | NOVEL SCREENING METHOD | HINUMA, SHUJI |
| <u>10498848</u> | Not Issued | 019 | 01/01/0001 | METHOD OF ANALYZING GENE EXPRESSION | HINUMA, SHUJI |
| <u>10488523</u> | Not Issued | 030 | 03/02/2004 | USE OF G PROTEIN-COUPLED RECEPTOR PROTEIN | HINUMA, SHUJI |
| <u>10488480</u> | Not Issued | 030 | 06/15/2004 | NOVEL SECRETORY PROTEINS AND DNA THEREOF | HINUMA, SHUJI |
| <u>10488038</u> | Not Issued | 030 | 02/27/2004 | SCREENING METHOD | HINUMA, SHUJI |
| <u>10487634</u> | Not Issued | 071 | 02/24/2004 | NOVEL RFRP-3 AND DNA THEREOF | HINUMA, SHUJI |
| <u>10480739</u> | Not Issued | 030 | 12/12/2003 | NOVEL LIGAND AND DNA THEREOF | HINUMA, SHUJI |
| <u>10460614</u> | Not Issued | 160 | 06/12/2003 | G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES | HINUMA, SHUJI |

| | | | | | |
|---------------------------------|------------|-----|------------|---|---------------|
| | | | | AND POLYNUCLEOTIDES | |
| <u>10332738</u> | Not Issued | 030 | 01/10/2003 | NOVEL POLYPEPTIDE AND DNA THEREOF | HINUMA, SHUJI |
| <u>10309870</u> | Not Issued | 160 | 12/04/2002 | G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES | HINUMA, SHUJI |
| <u>10278087</u> | Not Issued | 030 | 10/22/2002 | G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION, AND USE THEREOF | HINUMA, SHUJI |
| <u>10239321</u> | Not Issued | 030 | 09/19/2002 | PEPTIDE DERIVATIVES | HINUMA, SHUJI |
| <u>10221841</u> | Not Issued | 041 | 09/12/2002 | NOVEL MASS RECEPTOR-ANALOGOUS PROTEIN AND DNAS THEREOF | HINUMA, SHUJI |
| <u>10203015</u> | Not Issued | 030 | 09/23/2002 | SCREENING METHOD | HINUMA, SHUJI |
| <u>10185465</u> | Not Issued | 160 | 06/27/2002 | G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES | HINUMA, SHUJI |
| <u>10184722</u> | Not Issued | 030 | 06/27/2002 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HINUMA, SHUJI |
| <u>10184426</u> | Not Issued | 041 | 06/28/2002 | G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF | HINUMA, SHUJI |
| <u>10168048</u> | Not Issued | 030 | 06/12/2002 | NOVEL POLYPEPTIDE AND DNA THEREOF | HINUMA, SHUJI |
| <u>10148497</u> | Not Issued | 083 | 05/28/2002 | SCREENING METHOD | HINUMA, SHUJI |
| <u>10130584</u> | Not Issued | 041 | 05/16/2002 | USE OF PEPTIDE | HINUMA, SHUJI |
| <u>10111896</u> | Not Issued | 041 | 04/30/2002 | GHSR LIGAND POLYPEPTIDES AND DNAS THEREOF | HINUMA, SHUJI |
| <u>10090569</u> | Not Issued | 041 | 03/04/2002 | GALANIN RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF | HINUMA, SHUJI |
| <u>10069228</u> | Not Issued | 071 | 02/21/2002 | SCREENING METHOD | HINUMA, SHUJI |
| <u>10067477</u> | Not Issued | 161 | 02/04/2002 | G PROTEIN COUPLED RECEPTOR PROTEINS, THEIR PRODUCTION AND USE | HINUMA, SHUJI |
| <u>10044592</u> | Not Issued | 161 | 01/10/2002 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HINUMA, SHUJI |

| | | | | | |
|-----------------|------------|-----|------------|---|----------------|
| <u>09942367</u> | Not Issued | 160 | 08/29/2001 | G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES | HINUMA, SHUJI |
| <u>09929752</u> | Not Issued | 161 | 08/14/2001 | G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF | HINUMA, SHUJI |
| <u>09868885</u> | Not Issued | 041 | 06/22/2001 | USE OF PEPTIDE | HINUMA, SHUJI |
| <u>09787658</u> | Not Issued | 161 | 03/20/2001 | APJ LIGAND PEPTIDE DERIVATIVES | HINUMA, SHUJI |
| <u>09744226</u> | Not Issued | 161 | 01/22/2001 | NOVEL G PROTEIN COUPLED RECEPTOR AND ITS DNA | HINUMA, SHUJI |
| <u>09716147</u> | Not Issued | 092 | 11/17/2000 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HINUMA, SHUJI |
| <u>09700643</u> | Not Issued | 094 | 02/02/2001 | ANTIBODY AND USE THEREOF | HINUMA, SHUJI |
| <u>09576290</u> | 6794491 | 150 | 05/23/2000 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HINUMA, SHUJI |
| <u>09505396</u> | Not Issued | 161 | 02/16/2000 | RECEPTOR EXPRESSION CELLS AND USE THEREOF | HINUMA, SHUJI |
| <u>09481535</u> | 6180792 | 150 | 01/12/2000 | CONDENSED-RING THIOPHENE DERIVATIVES AND THIENOPYRIMIDE DERIVATIVES, THEIR PRODUCTION AND USE | HINUMA, SHUJI |
| <u>09461436</u> | 6538107 | 150 | 12/14/1999 | G PROTEIN COUPLED RECEPTOR PROTEIN PRODUCTION, AND USE THEREOF | HINUMA, SHUJI |
| <u>09446543</u> | Not Issued | 161 | 12/20/1999 | PROLACTIN SECRETION MODULATOR | HINUMA, SHUJI |
| <u>09403639</u> | Not Issued | 161 | 10/25/1999 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HINUMA , SHUJI |
| <u>09390799</u> | Not Issued | 161 | 09/07/1999 | NOVEL-HUMAN G-PROTEIN COUPLED RECEPTOR PROTEIN AND ITS DNA | HINUMA , SHUJI |
| <u>08423691</u> | 5677184 | 150 | 04/18/1995 | CHO CELLS THAT EXPRESS HUMAN LH-RH RECEPTOR | HINUMA , SHUJI |
| <u>08386354</u> | 5556946 | 150 | 02/08/1995 | INTERLEUKIN-2/VIRAL ANTIGEN PROTEIN CHIMERS | HINUMA , SHUJI |
| <u>08288663</u> | 5879896 | 150 | 08/09/1994 | A METHOD OF SCREENING FOR INHIBITORS OF HUMAN THYROTROPIN RELEASING HORMONE (TRH) RECEPTOR | HINUMA , SHUJI |

| | | | | | |
|-----------------|------------|-----|------------|---------------------------------------|----------------|
| <u>08181968</u> | Not Issued | 166 | 01/18/1994 | HUMAN INTERLEUKIN 2 | HINUMA , SHUJI |
| <u>08086429</u> | Not Issued | 164 | 06/30/1993 | ANTIGEN/INTERLEUKIN-2 FUSION PROTEINS | HINUMA , SHUJI |
| <u>07181362</u> | Not Issued | 166 | 04/13/1988 | ASSAY FOR ANTI-PRE-S ANTIBODY | HINUMA , SHUJI |

[Search and Display More Records.](#)

Search Another: Inventor

| | |
|---------------------------------------|------------------------------------|
| Last Name | First Name |
| <input type="text" value="HINUMA"/> | <input type="text" value="SHUJI"/> |
| <input type="button" value="Search"/> | |

To go back use Back button on your browser toolbar.

Back to [PALM](#) | [ASSIGNMENT](#) | [OASIS](#) | Home page

Day : Saturday
Date: 1/15/2005

Time: 16:08:14

PALM INTRANET**Inventor Name Search Result**

Your Search was:

Last Name = HOSOYA

First Name = MASAKI

| Application# | Patent# | Status | Date Filed | Title | Inventor Name 36 |
|---------------------------------|----------------|---------------|-------------------|--|-------------------------|
| <u>10719587</u> | Not Issued | 020 | 11/21/2003 | NOVEL G PROTEIN-COUPLED RECEPTOR PROTEIN, ITS DNA AND LIGAND THEREOF | HOSOYA,
MASAKI |
| <u>10504726</u> | Not Issued | 020 | 08/13/2004 | NOVEL SCREENING METHOD | HOSOYA,
MASAKI |
| <u>10488523</u> | Not Issued | 030 | 03/02/2004 | USE OF G PROTEIN-COUPLED RECEPTOR PROTEIN | HOSOYA,
MASAKI |
| <u>10488480</u> | Not Issued | 030 | 06/15/2004 | NOVEL SECRETORY PROTEINS AND DNA THEREOF | HOSOYA,
MASAKI |
| <u>10488038</u> | Not Issued | 030 | 02/27/2004 | SCREENING METHOD | HOSOYA,
MASAKI |
| <u>10487634</u> | Not Issued | 071 | 02/24/2004 | NOVEL RFRP-3 AND DNA THEREOF | HOSOYA,
MASAKI |
| <u>10474481</u> | Not Issued | 030 | 10/08/2003 | SCREENING METHOD | HOSOYA,
MASAKI |
| <u>10239321</u> | Not Issued | 030 | 09/19/2002 | PEPTIDE DERIVATIVES | HOSOYA,
MASAKI |
| <u>10203015</u> | Not Issued | 030 | 09/23/2002 | SCREENING METHOD | HOSOYA,
MASAKI |
| <u>10184722</u> | Not Issued | 030 | 06/27/2002 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HOSOYA,
MASAKI |
| <u>10168048</u> | Not Issued | 030 | 06/12/2002 | NOVEL POLYPEPTIDE AND DNA THEREOF | HOSOYA,
MASAKI |
| <u>10148497</u> | Not Issued | 083 | 05/28/2002 | SCREENING METHOD | HOSOYA,
MASAKI |
| <u>10090569</u> | Not Issued | 041 | 03/04/2002 | GALANIN RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF | HOSOYA,
MASAKI |
| <u>10069228</u> | Not Issued | 071 | 02/21/2002 | SCREENING METHOD | HOSOYA,
MASAKI |
| <u>10067477</u> | Not | 161 | 02/04/2002 | G PROTEIN COUPLED | HOSOYA, |

| | | | | | |
|-----------------|------------|-----|------------|--|-----------------|
| | Issued | | | RECEPTOR PROTEINS, THEIR PRODUCTION AND USE | MASAKI |
| <u>09935371</u> | Not Issued | 161 | 08/22/2001 | PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF | HOSOYA, MASAKI |
| <u>09831758</u> | Not Issued | 161 | 05/11/2001 | NOVEL G PROTEIN COUPLED RECEPTOR PROTEIN ITS DNA AND LIGAND THEREOF | HOSOYA, MASAKI |
| <u>09716147</u> | Not Issued | 092 | 11/17/2000 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HOSOYA, MASAKI |
| <u>09576290</u> | 6794491 | 150 | 05/23/2000 | POLYPEPTIDES, THEIR PRODUCTION AND USE | HOSOYA, MASAKI |
| <u>09261085</u> | 6113960 | 150 | 03/02/1999 | METHOD FOR COATING FOOD PIECES WITH POWDER MATERIAL | HOSOYA , MASAKI |
| <u>09255518</u> | 6492324 | 150 | 02/22/1999 | APJ LIGAND POLYPEPTIDES | HOSOYA , MASAKI |
| <u>09201474</u> | 6399316 | 150 | 11/30/1998 | PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF | HOSOYA , MASAKI |
| <u>09038572</u> | Not Issued | 161 | 03/11/1998 | G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION, AND USE THEREOF | HOSOYA , MASAKI |
| <u>08978525</u> | Not Issued | 161 | 11/25/1997 | DNA PRIMER AND A METHOD FOR SCREENING DNAs | HOSOYA , MASAKI |
| <u>08932325</u> | 6058877 | 150 | 09/17/1997 | APPARATUS FOR SPRINKLING FOOD PIECES WITH POWDER MATERIAL | HOSOYA , MASAKI |
| <u>08856154</u> | 5973117 | 150 | 05/14/1997 | PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF | HOSOYA , MASAKI |
| <u>08855213</u> | 5892004 | 150 | 05/13/1997 | METHOD FOR PREPARING PACAP RECEPTOR PROTEIN | HOSOYA , MASAKI |
| <u>08811897</u> | 5858787 | 150 | 03/05/1997 | DNA ENCODING PACAP RECEPTOR PROTEIN AND METHOD FOR PREPARING SAID PROTEIN | HOSOYA , MASAKI |
| <u>08796570</u> | Not Issued | 161 | 02/06/1997 | G PROTEIN COUPLED RECEPTOR PROTEINS, THEIR PRODUCTION AND USE | HOSOYA , MASAKI |
| <u>08776971</u> | 6228984 | 150 | 02/07/1997 | POLYPEPTIDES THEIR | HOSOYA , |

| | | | | PRODUCTION AND USE | MASAKI |
|-----------------|----------------|-----|------------|---|-----------------|
| <u>08566037</u> | <u>5756295</u> | 150 | 12/01/1995 | DNA PRIMER AND A METHOD FOR SCREENING DNAs | HOSOYA , MASAKI |
| <u>08550120</u> | <u>5985554</u> | 150 | 10/30/1995 | METHOD OF PROVING THE FUNCTION OF PROTEINS OR PEPTIDES ENCODED BY PARTIALLY SEQUENCED CDNAs BY INHIBITING PROTEIN SYNTHESIS WITH ANTISENSE OLIGONUCLEOTIDES | HOSOYA , MASAKI |
| <u>08540650</u> | <u>6399325</u> | 150 | 10/11/1995 | GALANIN RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF | HOSOYA , MASAKI |
| <u>08513974</u> | <u>6114139</u> | 150 | 09/14/1995 | A G- PROTEIN COUPLED RECEPTOR PROTEIN AND A DNA ENCODING THE RECEPTOR | HOSOYA , MASAKI |
| <u>08288663</u> | <u>5879896</u> | 150 | 08/09/1994 | A METHOD OF SCREENING FOR INHIBITORS OF HUMAN THYROTROPIN RELEASING HORMONE (TRH) RECEPTOR | HOSOYA , MASAKI |
| <u>08202986</u> | Not Issued | 166 | 02/25/1994 | PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF | HOSOYA , MASAKI |

Inventor Search Completed: No Records to Display.

Search Another: Inventor

Last Name

First Name

To go back use Back button on your browser toolbar.

Back to [PALM](#) | [ASSIGNMENT](#) | [OASIS](#) | Home page